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Sellers, R.,[1,2] Harold, G.T.,[1,2,3] Smith, A. F.,[1] Neiderhiser, J.M.,[4] Reiss, D.,[5] Shaw, D.,[6] Natsuaki, M.N.,[7] Thapar, A.,[2] Leve, L.D.[8]

[1] School of Psychology, Andrew and Virginia Rudd Centre for Adoption Research and Practice, University of Sussex, Brighton, UK

[2] Institute of Psychological Medicine and Clinical Neurosciences, School of Medicine, MRC Centre for Neuropsychiatric Genetics and Genomics, Cardiff University, Cardiff, UK

[3] School of Psychology, Trinity College Dublin, The University of Dublin, Ireland

[4] Department of Psychology, The Pennsylvania State University, University Park, PA, USA

[5] Yale Child Study Center, New Haven, CT, USA

[6] University of Pittsburgh, Pittsburgh, PA, USA

[7] University of California, Riverside, CA, USA

[8] Prevention Science Institute, University of Oregon, Eugene, USA

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Corresponding Author: Gordon T Harold, School of Psychology, Andrew and Virginia Rudd Centre for Adoption Research and Practice, University of Sussex, Brighton.

Phone: +44 (0)1273 877102

E*mail*: G.Harold@sussex.ac.uk

Abstract

Background: Attention Deficit Hyperactivity Disorder (ADHD) is highly heritable and is associated with lower educational attainment. ADHD is linked to family adversity, including hostile parenting. Questions remain regarding the role of genetic and environmental factors underlying processes through which ADHD symptoms develop and influence academic attainment.

Method: This study employed a parent-offspring adoption design (*N*=345) to examine the interplay between genetic susceptibility to child attention problems (birth mother ADHD symptoms) and adoptive parent (mother and father) hostility on child lower academic outcomes, via child ADHD symptoms. Questionnaires assessed birth mother ADHD symptoms, adoptive parent (mother and father) hostility to child, early child impulsivity/activation, and child ADHD symptoms. The Woodcock-Johnson test was used to examine child reading and math aptitude.

Results: Building on a previous study (Harold et al., 2013), heritable influences were found: birth mother ADHD symptoms predicted child impulsivity/activation. In turn, child impulsivity/activation (4.5 years) evoked maternal and paternal hostility, which was associated with children's ADHD continuity (6 years). Both maternal and paternal hostility (4.5 years) contributed to impairments in math but not reading (7 years), via impacts on ADHD symptoms (6 years).

Conclusion: Findings highlight the importance of early child behavior dysregulation evoking parent hostility in both mothers *and* fathers, with maternal and paternal hostility contributing to the continuation of ADHD symptoms and lower levels of later math ability. Early interventions may be important for the promotion of child math skills in those with ADHD symptoms, especially where children have high levels of early behavior dysregulation.

Keywords: ADHD, hostile parenting, reading, math, gene-environment correlation

Disentangling Nature from Nurture in Examining the Interplay Between Parent-Child Relationships, ADHD, and Early Academic Attainment

ADHD is a childhood-onset neurodevelopmental disorder characterized by symptoms of hyperactivity-impulsiveness and inattention (American Psychological Association, 1994). Early markers of behavioral and emotional dysregulation (e.g., impulsivity, reactivity, regulation difficulties) in infancy and childhood have been associated with increased risk for ADHD (Harold et al, 2013; Frick et al 2018). ADHD is highly heritable, with twin studies estimating the heritability of ADHD to be around 70% (Thapar et al., 2018). Recent molecular genetics studies have also evidenced the genetic underpinnings of ADHD (DeMontis et al., 2018). However, the rearing environment, including parenting and parent-child relationship quality, is also recognized as important with respect to modifying the course of ADHD symptoms in children (Deault, 2010; Johnston & Mash, 2000), even when genetic factors are considered (Thapar et al., 2006; Thapar et al., 2013).

A number of studies have found associations between symptoms and diagnosis of ADHD and lower academic attainment in childhood, adolescence, and adulthood (Daley & Birchwood, 2010; Greven et al., 2014; Greven et al., 2011; Plourde et al., 2015; Snowling & Hulme, 2012; Tosto et al., 2015). Specifically, ADHD symptoms have been associated with reduced reading, writing, and math attainment in both clinical and community samples (Daley & Birchwood, 2010). Associated comorbid specific learning problems (e.g., dyslexia, developmental coordination disorder) and poorer cognitive ability that are known to be strongly associated with ADHD may be one route via which ADHD impacts on educational attainment. The pathways and processes through which ADHD symptomology may influence academic achievement are not clear. Genetic and environmental pathways are both hypothesized to play a role in these associations (Hart et al., 2010; Kuntsi et al., 2004; Paloyelis et al., 2010; Willcutt et al., 2007).

Parenting behaviors have been associated with child ADHD symptoms (Johnston & Mash, 2001); the most consistent finding is an association with hostile parenting (Harold et al., 2013; Ullsperger, Nigg, & Nikolas, 2016). Recent twin and adoption studies also suggest that behaviours consistent with ADHD symptoms in the early years of life (e.g., child impulsivity/activation) may evoke hostile parenting (Harold et al., 2013; Lifford et al, 2009). Parenting processes, specifically hostile caregiving, also have been associated with reduced academic attainment, including reading and math performance in middle childhood and adolescence (Flouri and Buchanon, 2004; Eamon, 2005; Benner & Kim, 2010; Dotterer et al., 2008; McCoy et al., 2013; Melby & Conger, 1996; Weymouth et al., 2016), with initial evidence suggesting that youth adjustment may play a mediating role (e.g., Wentzel, 1994). However, there is relatively limited examination of the parenting processes that may impact on ADHD outcomes (Deault, 2010). Furthermore, when examining the role of the parent-child relationship on child adjustment, research has primarily focused on the mother-child relationship. The role of fathers is increasingly recognized as an important influence on risk for child psychopathology and academic attainment (e.g., Cabrera et al., 2018). Although evidence suggests that associations between parenting and child outcomes may vary across the mother-child and father-child relationship (Harold et al., 2013; Lifford et al., 2009), this is rarely considered in the context of child ADHD symptoms. Where studies examine maternal and paternal parenting processes in relation to child ADHD symptoms, they have not usually considered the *relative* role of maternal and paternal parenting processes (Lifford, Harold, & Thapar, 2008; Kaiser et al., 2011).

A key challenge to establishing family processes as a salient environmental factor for child outcomes is that most family studies rely on genetically related parents and children and therefore it is difficult to disentangle environmental from genetic effects, also known as gene-environment correlations (*r*GE; Plomin, DeFries & Loehlin, 1977; Harold, Leve & Sellers,

2017). The two most frequently examined forms of *r*GE are passive and evocative *r*GE. Passive *r*GE occurs when parents' and children's genes (which are shared) confound the association between family and child level variables (Plomin et al., 1977; Scarr & McCartney 1983). Evocative *r*GE occurs when genetically influenced characteristics in the child evoke particular responses from their environment (Plomin et al., 1977). Environmental main effects are those that cannot be attributed to *r*GE. To disentangle such effects, genetically informative designs, among others (Thapar & Rutter, 2019) are helpful for attempting to identify likely causal processes to target intervention and prevention strategies appropriately. To address this limitation, we utilized a longitudinal parent-offspring adoption design to examine the interplay between genetic susceptibility to child attention problems (birth mother ADHD symptoms) and adoptive parent (mother and father) hostility on child academic outcomes. This design examines the association between adopted children symptoms and characteristics of biologically related parents (the birth parents) and unrelated parents (the adoptive parents). Associations between adopted children and their adoptive parents are assumed to be due to rearing environmental influences, unconfounded by shared genes (i.e., removing the confound of passive *r*GE). In contrast, associations between adopted children and their biological parents are assumed to be attributable to shared genes (in addition to prenatal environmental effects for biological mothers). Employing this research design, we examined associations between adoptive mother and father hostile parenting (at child age 4.5 years), child ADHD symptoms (child age 6 years), and academic attainment (reading and math scores at age 7 years). We also examined associations between genetically influenced (via birth mother symptoms of ADHD) early child behaviors (impulsivity/activation, behavior consonant with early ADHD-type behaviors: Harold et al., 2013) on both maternal and paternal hostility (i.e., evocative *r*GE) as pathways associated with child ADHD symptom continuity, reading, and math achievement. It was hypothesized that biological

mother ADHD symptoms would be associated with adopted child impulsivity/activation (at age 4.5 years), which in turn would predict maternal and paternal hostility, indicative of evocative rGE, replicating prior findings for mothers. Additionally, it was hypothesized that maternal and paternal hostile parenting (at age 4.5 years) would predict children's ADHD symptom continuity at age 6 years and that child ADHD symptoms would predict poorer academic achievement.

Methods

Participants and study design

The current sample comprised 361 linked sets of adopted children, adoptive parents, and biological mothers from the Early Growth and Development Study (EGDS), a longitudinal, multisite US parent-offspring adoption study (Leve et al., 2019). The median lag-time to placement was child age of 2 days (M = 6.2; SD = 12.45; range = 0-91 days). Participants were representative of the adoptive parent and birth parent populations that completed adoption plans at the participating agencies during the same period. Mean age of birth mothers and birth fathers at the time of placement was 23.84 and 25.61 respectively. The majority of birth parents were Caucasian (birth mother = 75%; birth father = 79%). Birth parents typically had high school education, and household incomes of <$25,000. The majority of adoptive parents were Caucasian (91%). Mean age of adoptive mothers and adoptive fathers was 36.98 and 37.82 at the time of placement, respectively. Adoptive parents were typically college educated with a median household income of $100,000. Adoptive parents had been married an average of 12 years. Additional details about the study design and sample description are described elsewhere (Leve et al., 2019; Leve et al., 2007). Ethical approval was provided by the University of Oregon Institutional Review Board (protocol number: 04262013.036). Given our focus on maternal relative to paternal parenting

processes, we excluded same-sex couples from analyses, therefore 345 families were available for the current analysis.

Measures

Birth Mother ADHD symptoms

Birth mother ADHD symptoms were assessed using maternal reports of both the Adult Temperament Questionnaire (ATQ; α = .73) at 18 months of child age (Rothbart, Ahadi, & Evans, 2000) and the Barkley Adult ADHD scale (α = .90) at child age 4.5 years (Murphy & Adler, 2004). The scales were standardized and then summed into a single measure of mother ADHD symptoms, with good internal consistency (α = .88). See Harold et al, 2013 for further details of the measure (*M* = .08, *SD* = 1.75).

Adoptive parent-to-child hostility

Adoptive parent-to-child hostility was assessed using parent self-reports on the Iowa Family Interaction Rating Scales (Melby et al., 1993) at child age 4.5 years. Adoptive mothers and fathers reported on their own hostile behaviors towards their child (maternal reports *M* = 11.04, *SD* = 3.08; paternal reports *M* = 10.29; *SD* =2.89). Higher scores were indicative of higher hostility (maternal reports α = .91; paternal reports: α = .94).

Child Impulsivity/Activation

Child Impulsivity/Activation was assessed using adoptive mother report on the Children's Behavior Questionnaire: CBQ (Rothbart, Ahadi, Hershey, & Fisher, 2001) and adoptive mother reports on the Behavioral Inhibition Scale/Behavioral Activation Scale: BIS/BAS (Blair, Peters, & Granger, 2004) at age 4.5 years, as used in Harold et al. (2013) . Each of these subscales were standardized and then summed to create a single indicator of early child impulsivity/ activation (*M* = .02, *SD* = 3.19).

Child ADHD symptoms

Child ADHD symptoms were assessed using adoptive mother and father reports on the Conner's Abbreviated Parent Questionnaire, a 10-item scale regarding hyperactivity and inattentive behaviors (Conners, 1997) at child age 6 years. A composite score of mother and father reports was created (r = .71, p <.001), using a mean score of mother and father reports (M = 8.09; SD =4.93).

Child academic achievement

Child academic achievement was measured at age 7 years using z-scores of reading (M = .53, SD =1.00) and math fluency (M = -.02; SD = 1.04) subscales from the Woodcock-Johnson III achievement test (Woodcock, McGrew, & Mather, 2001), which assessed reading and math skills. Previous research suggests that the reliability for both reading and math fluency show strong reliabilities of .90 (Schrank & McGrew, 2001). Scales were reverse scored so that higher scores indicated greater levels of difficulty in attaining competence (i.e., poorer academic attainment).

Covariates

Earlier levels of academic achievement were assessed using the Test of Preschool Early Literacy test (TOPEL) at age 4.5 years (Lonigan, Wagner, & Togesen, 2007). The present study combined two subscales (Print Knowledge and Definitional Vocabulary) to create a composite score of children's emergent literacy skills. Previous research suggests internal consistency for the TOPEL index is .96 (Lonigan et al., 2007).

To control for similarities between birth and adoptive families resulting from contact and knowledge between birth parents and children, secondary analyses considered the association with the level of openness in the adoption (Ge et al., 2008). We also examined associations with prenatal complications to attempt to disentangle genetic influences from prenatal environment (Marceau et al., 2016). However, neither of these covariates was

significantly associated with any variables in the model and therefore neither was considered further in analyses.

Analyses

Path analysis was used to examine the role of biological mother ADHD as a predictor of adopted children's early impulsivity/activation behaviors, and to examine associations between child impulsivity/activation and both adoptive mother and father hostility, thereby allowing examination of evocative *r*GE processes. It simultaneously allowed the examination of processes through which parental hostility and child ADHD symptoms may be associated with later child academic outcomes (reading and math). The full theoretical model is shown in Figure 1. Analyses were conducted using LISREL (Joreskog & Sorbom, 2006). Fit statistics were used to examine model fit using the chi square, Confirmatory Fit Index (CFI), and the Root Mean Square Error of Approximation (RMSEA). Good model fit is indicated by a non-significant chi square test, CFI $\geq$ .98, TLI $\geq$ .80, and RMSEA $\leq$ .05 (Kline, 2005).

Missing data ranged from 21% (72/345 for math achievement at age 7) to 34% (117/345 father hostility at age 4.5 years). The Little's test indicated that data were missing completely at random (χ^2 (253) = 281.54, p = .105). Analyses were conducted using Full Information Maximum Likelihood estimation, which makes use of all available data, therefore 345 cases were included in the current analyses.

Results

Correlational analyses

As previously demonstrated (Harold et al., 2013), birth mother ADHD symptoms were correlated with early child impulsivity/activation (r = .18, p <.001); see Table 1. In addition, early child impulsivity was correlated with maternal and paternal hostility toward the child (r = .20, p <.001; r = .21, p <.001 respectively), as well as later child ADHD symptoms (r = .42, p <.001), but not with later reading (r =.09, p =.184) or math scores (r =

.05, *p* >.250). Maternal and paternal hostility toward the child were correlated with each other (*r* = .33, *p* <.001), and with later levels of child ADHD symptoms (*r* = .27, *p* <.001; *r* = .26, *p* <.001 respectively). Parent hostility was not correlated with child reading (maternal hostility: *r* = .10, *p* =.138; paternal hostility: *r* = .12, *p* =.107) or math scores (maternal hostility: *r* = -.01, *p* >.250; paternal hostility: *r* = .08, *p* = .245). Child ADHD symptoms were correlated with lower math (*r* = .22, *p* <.001) but not reading achievement (*r* = .11, *p* =.118).

Path analysis

Figure 1 shows the full model. Fit indices indicated a satisfactory fit to the data (χ^2 (9) = 26.66, p = .002; CFI = .96; TLI = .87; RMSEA = .07 (.04, .10), SRMR = .05).

[Figure 1]

Birth mother ADHD symptoms predicted child early impulsivity/activation (β = .17, *p* = .005). Child impulsivity/activation (at age 4.5 years) in turn predicted both adoptive mother and father hostility (β =.20, *p* = .002; β = .21, *p* = .001 respectively), as well as child ADHD symptoms at age 6 years (β = .35, *p* <.001). Adoptive mother hostility predicted later child ADHD symptoms (β = .13, *p* = .015), as did adoptive father hostility (β = .16, *p* = .006). Neither maternal hostility nor father hostility directly predicted later poorer child math (β =-.10, *p* = .093; β =-.03, *p* >.250) or reading (β = .03, *p* >.250; β = .04, *p* >.250) achievement. However, there was a significant indirect effect of maternal and paternal hostility on later math aptitude via the continuity in child ADHD symptoms (both β = .03, *p* <.05). Early levels of poorer reading in the child predicted later poorer math (β = .29, *p* <.001) and reading (β = .46, *p* <.001) achievement. Child symptoms of ADHD predicted later poorer child math achievement (β = .16, *p* = .007), but not reading (β = -.03, *p* >.250). There was a significant indirect effect of early child impulsivity/activation on later child poorer math ability via child ADHD symptoms (β = .05, *p* <.05). Additional analyses compared this full model to a model which set non-significant associations to zero. A non-significant chi-square difference test

(Δdf = 5; $\Delta\chi$2 = 6.16, p=.292) suggested that these non-significant associations did not substantially contribute to the model indicating that a more parsimonious model can be accepted.

Stacked modelling procedures examined whether pathways from child impulsivity to maternal and paternal hostility differed in magnitude. Constraining the path from child impulsivity to adoptive mother hostility and to adoptive father hostility to be equal did not result in a significantly worse model fit (Δdf = 1; $\Delta\chi^2$ = 0.98, p > .05), suggesting that the association between early child impulsivity on father hostility was not significantly different than its association with mother hostility. We also examined whether pathways differed in magnitude between maternal and paternal hostility to child ADHD symptoms. Constraining the path from adoptive mother hostility and father hostility to child ADHD symptoms to be equal did not result in a significantly worse model fit (Δdf = 1; $\Delta\chi^2$ = .78, p > .05), suggesting that the associations between maternal and paternal hostility on child ADHD symptoms was not significantly different.

Discussion

The current study examined the interplay between genetic susceptibility and both maternal and paternal hostility in the persistence of ADHD symptoms and academic achievement in childhood with child impulsivity/activation as an early marker of risk for ADHD symptoms (Frick et al., 2018). We examined associations between early child attributes (impulsivity/activation), parent hostility and academic attainment (specifically math) via child ADHD symptoms in a research design that removes the confound of passive *r*GE (adoptive parents and their adopted children do not share genes). Our findings first replicate previous findings in this sample suggesting that genetically influenced (measured by birth mother ADHD symptoms) early child impulsivity/activation evoke adoptive mother hostility (Harold et al., 2013). We also extended findings, demonstrating evocative effects on

adoptive father hostility. Second, child ADHD symptoms were associated with later academic attainment. We found that child ADHD symptoms were specifically associated with lower math but not reading attainment. This is surprising given that ADHD and reading ability are known to be strongly associated (e.g., Willcutt et al., 2007; Snowling & Hulme, 2012; Adams & Snowling, 2001), with evidence suggesting shared genetic liability (Plourde et al., 2015; Stergiakouli et al., 2017; Barry, Lyman, & Klinger, 2002; Daley & Birchwood, 2010). The null finding on ADHD symptoms and reading is in contrast to previous research that finds associations between ADHD and reading/literacy (Rabiner, Coie, & Conduct Problems Prevention Group, 2000; Frazier, Youngstrom, Glutting & Watkins, 2007). There are several explanations for why the current study found no association between child ADHD symptoms and reading. First, the current sample consists of primarily middle-class, college-educated adoptive parents and evidence suggest that parents with higher educational qualifications read to their children more frequently (Kuo, Franke et al, 2004). Second, parents place an increased importance on literacy learning and spend more time supporting children's reading compared to math (Cannon & Ginsberg, 2008). This results in math being a relatively novel subject at school entry. Third, we were able to control for prior reading abilities using the TOPEL, but we did not have an earlier measure of children's math abilities. This methodological artefact may have resulted in more available variance to predict in math as compared to reading achievement. Together, these three factors could have contributed to reading being less influenced by ADHD symptoms in the current study. Nevertheless, these findings have implications for the understanding of child development and long-term math attainment where there are indicators of signs of early ADHD.

We were able to examine pathways implicated in the development of ADHD symptomatology and subsequent academic attainment in a study that removed the confound of passive *r*GE. Any associations between adoptive parent characteristics and adopted

children cannot be explained by common genes, and likely reflect environmental associations, providing evidence of the importance of environmental factors, specifically parent hostility for child ADHD symptom continuity, and later math attainment. In line with previous research, we found evidence of an association between child ADHD symptoms and maternal hostility (e.g., Harold et al., 2013); however, we extend current understanding, demonstrating the importance of the relative role of father hostility for child ADHD symptom persistence. The current study suggests that the associations between parental hostility and later child ADHD symptoms do not differ in magnitude for mothers or fathers. This has implications for recognizing the potential of father involvement in interventions. In addition, we were also able to examine evocative *r*GE, specifically examining genetically informed attributes of the child (impulsivity/activation) on both maternal and paternal parenting processes. Consistent with previous research examining evocative *r*GE, early impulsivity/activation evoked hostile parenting in mothers with maternal hostility in turn predicting later ADHD symptoms (Harold et al., 2013). We found similar processes for paternal parenting, with child impulsivity/activation evoking hostile paternal parenting, which in turn predicted later child ADHD symptoms. These findings have implications for the understanding of child development, with both genetic (measured by birth mother ADHD symptoms) and environmental processes (adoptive mother and father hostile parenting) predicting child outcomes (child ADHD symptoms and later math attainment).

Limitations and future directions

It is important to interpret these findings in the light of limitations. First, potential bidirectional effects between early impulsivity/activation and parent hostility may be present, however, in the current study, early child impulsivity/activation was measured at the same period as adoptive parent hostility. However, findings from the current study are consistent with a number of studies that have found evidence of evocative *r*GE for parenting behaviors

using molecular genetic (Elam et al., 2016) and family-based genetic research designs (Elam et al., 2014; Harold et al., 2013). Further research is needed to examine the direction of effects between early impulsivity and parenting processes. Second, birth father ADHD symptoms were not included in the measure of the child's inherited risk because of limited data available from birth fathers. ADHD diagnoses are more common in males than females (Polanczyk, de Lima, Horta, Biederman, & Rohde, 2007). We also relied on adult self-reports of ADHD symptoms rather than diagnoses of ADHD in birth mothers as an index of genetic risk for impulsivity in childhood. Together, these factors may have underestimated (or overestimated) the magnitude of the association between birth parent ADHD symptoms and child impulsivity. Third, although the findings in the current study are consistent with Harold and colleagues (2013) which examined associations between adoptive mother hostility and child ADHD symptoms using a cross-rater approach, it is important to note that, for some measures there was a reliance on a single reporter (adoptive mother). Where available, we utilized multiple informants to reduce the potential risk for shared method variance, for example, both mother and father reports of child ADHD symptoms were used. We also used standardized assessments of child reading and math achievement. However, future research should consider alternative approaches to assessing child achievement, symptoms, and parenting (e.g., teacher reports or clinical assessments of child ADHD symptoms; observational parenting assessments). Fourth, in the current study, child ADHD symptom levels were relatively low and therefore do not necessarily constitute mental health 'difficulties'. However, ADHD can be conceptualized as a continuum: both in terms of associated outcomes, and because heritability estimates have been shown to be similar across the continuum as well as in 'high scores' (Stergiakouli et al., 2015). Therefore, non-clinical samples can be useful to examine the etiology of ADHD and related outcomes. Fifth, whilst both inattentive and hyperactive-impulsive symptoms of ADHD have been evidenced to

contribute to the prediction of reading and math, evidence suggests that inattentiveness may be a significantly stronger predictor of reading and math than hyperactive-impulsive symptoms of ADHD (e.g., Greven et al., 2011; Greven et al., 2014). Therefore, it is possible that different domains of ADHD may differentially impact on child reading and math outcomes. However, in the current study, child ADHD symptoms were assessed using the Conners' Parent Rating Scale – Revised, a unidimensional measure of ADHD symptoms. Therefore it was not possible to examine these domains separately.

Finally, ADHD commonly co-occurs with other neurodevelopmental problems (e.g. language, motor co-ordination difficulties; DuPaul et al. 2013; Martin et al., 2015) as well as mental health problems (e.g. conduct disorder, anxiety; Jensen et al., 1997; Thapar & van Goozen, 2018; Schatz & Rostain, 2006) so we cannot rule out that the link between ADHD and parent hostility and its links with math attainment is explained by these factors.

To further understand the pathways and processes influencing the developmental course of ADHD symptomatology and academic outcomes, future research should consider additional mediators and moderators of the pathways to child ADHD symptoms and academic outcomes. For example, additional aspects of parenting that were included in the current study but not in the current report (e.g., monitoring, engagement) could also be important for the development of child mental health difficulties (including symptoms of ADHD) and later academic functioning (Daley & Birchwood, 2010; Rogers et at., 2009a, 2009b). In addition, parent academic ability is associated with child academic ability, with the association due to both genetic and environmental influences (Friend et al., 2009). Future research should examine how adoptive and birth parent measures of academic ability impact on these processes. In addition, adoptive parent mental health may be important to consider: parental symptoms of ADHD have been associated with aspects of parenting (Harvey, Danforth, McKee, Ulaszek, & Friedman, 2003), as have symptoms of antisocial behaviour

(Harold et al., 2012; Harold et al., 2011). Therefore, it will be important for future research to examine how other aspects of parental mental health affect the processes through which children develop psychopathology, and difficulties in reading and math achievement.

Notwithstanding these caveats, results provide evidence of an environmental effect of adoptive father-to-child and mother-to-child effects on child ADHD symptom continuity, and later math ability in children to whom they were not genetically related (i.e., removing passive *r*GE). In addition, adoptive mother-to-child *and* father-to-child hostility was evoked by genetically informed child impulsivity/activation which also predicted child ADHD symptoms. The current data help advance understanding of the interplay between genetic susceptibility and environmental risk in the development of ADHD symptoms and academic achievement in childhood. Genetic risk for ADHD symptoms served as a risk factor for disrupted mother-to-child and father-to-child relationships. These findings suggest a cascade of risk through which genetic risk for ADHD symptoms influence later math achievement, with indirect effects via both mother and father hostility contributing to the developmental course of ADHD. Early interventions targeting hostility in *both* parents may be important, especially where children have high levels of impulsivity, not for treating ADHD per se (as per NICE guidance) but for influencing its developmental course and associated outcomes and attainment. There is evidence that parenting programs can be an effective intervention for those with ADHD who have comorbid conduct disorder (NICE 2018), which is known to also be associated with learning problems (Erskine et al., 2016). Recent review evidence suggests that parenting interventions targeting ADHD alone (without comorbid presence of conduct disorder) do not appear efficacious (Lange et al, 2018; Daley et al, 2018; see meta-analysis Sonuga-Barke et al., 2013) but may help other outcomes. Notwithstanding these observations, the present results are among the very first in this area to highlight the role of maternal and paternal parenting as significant in relation to ADHD symptoms - a potential

consideration for future intervention program design and development. In addition, child ADHD symptoms were associated with later academic ability, specifically math ability. Interventions that assist with the development of math skills, particularly those with high levels of early manifestations of ADHD symptoms, may also be particularly beneficial.

Author contributions

Harold, Leve, and Sellers developed the study research question. Sellers performed the data analysis under the supervision of Harold and Leve. Sellers and Harold contributed to the interpretation of the study findings. Sellers drafted the manuscript. Reiss, Leve, Neiderhiser, Shaw and Natsuaki designed and carried out the original study and data collection activities All authors provided critical revisions, and all authors approved the final version of the manuscript for submission.

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Table 1. Correlations, means, and standard deviations among study variables

	1	2	3	4	5	6	7
1. Birth mother ADHD symptoms [a]	--						
2. Child impulsivity [a]	**.18** **p.012**	--					
3. Maternal hostility	-.02 p>.250	**.20** **p.002**	--				
4. Paternal hostility	-.11 p.133	**.21** **p.002**	**.33** **p<.001**	--			
5. Child ADHD	.05 p>.250	**.42** **p<.001**	**.27** **p<.001**	**.26** **p<.001**	--		
6. Child lower reading aptitude [b]	-.02 p>.250	.09 p.184	.10 p.138	.12 p.107	.11 p.118	--	
7. Child lower math aptitude [b]	-.07 p>.250	.05 p>.250	-.01 p>.250	.08 p.245	**.22**[**] **p.001**	**.53**[*] **p<.001**	--
Mean (SD)	.08 (1.75)	.02 (3.19)	11.04 (3.08)	10.29 (2.89)	8.09 (4.93)	.53 (1.00)	-.02 (1.04)

[a] Summed standardized subscales; [b] Standardized z score

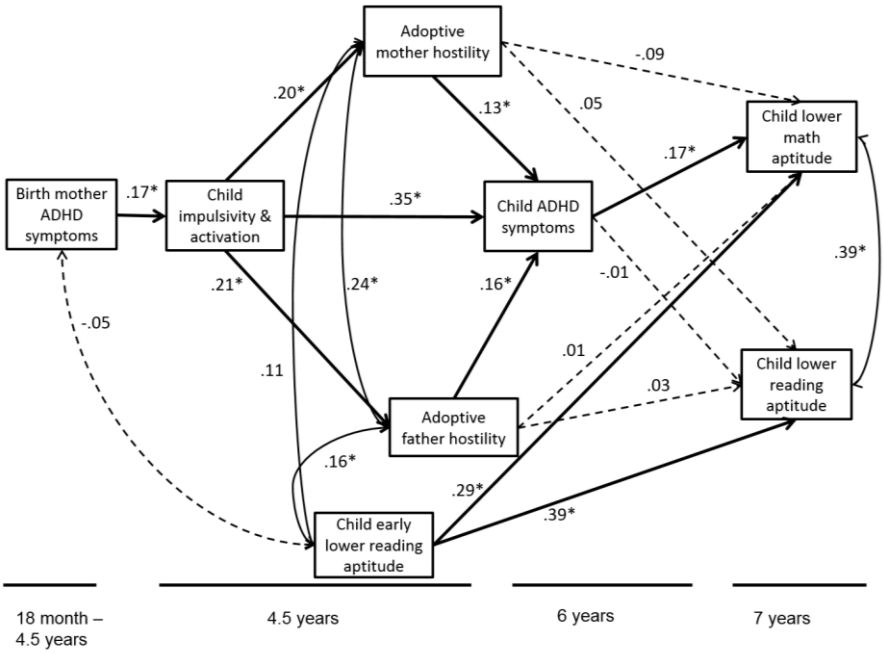

Fig. 1. Path model with results showing standardized coefficients

Path diagram shows standardized coefficients; * *p* = <.05; Solid lines represent significant coefficients; Dashed lines represent non-significant coefficients. Fit indices (Kline, 2011) indicated a satisfactory fit to the data: χ^2 (9) = 26.66, p = .002; CFI = .96; TLI= .87; RMSEA = .07 (.04, .10), SRMR = .05.